Relationship between Periodontitis and Preterm labour - Correlation with C Reactive protein levels

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CERTIFICATE

This is to certify that the dissertation titled "**Relationship between Periodontitis and Preterm labour - Correlation with C Reactive protein levels ”** is the bonafide work done by **Dr. KRIPA MOHAN** between April 2007 to April 2008 during her M.D.,O.G., course at ISO -KGH, MMC Chennai.

DEAN
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DIRECTOR
ISO-GOVT. KGH

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ETHICAL COMMITTEE CERTIFICATE

No:

Dated:

I, **Dr. KRIPA MOHAN** apply for the ethical committee certificate for the project **"RELATIONSHIP BETWEEN PERIODONTITIS AND PRETERM LOW BIRTH WEIGHT INFANTS"** under the guidance of **Dr. Prof. VASANTHA N. SUBBAIAH, Director, Institute of Social Obstetrics and Gynaecology in Govt K.G. Hospital, Chennai-600 005**

I understand the implications of doing research with human subjects and will fully comply with the regulations and keep the dignity and protect the health of subjects at all costs.

Signature of the Postgraduate Student

I have no objective to guiding this postgraduate student in the project mentioned above. I shall supervise to the extent that all the human rights are protected and research is carried on with utmost humanitarian principles

Signature of the Guide

Director of Social Obstetrics,
Institute of Social Obstetrics and
Govt. Kasturba Gandhi Hospital
For Women & Children
Triplicane, Chennai-600 005

Seal of Guide

I certify that this project has been presented in front of the Ethical Committee on duly formatted in this institution and that all the members of the ethical committee have given permission to conduct this research

CHAIRMAN ETHICAL COMMITTEE

Date:

Professor & Head
Dept. of Community Medicine
Stanley Medical College
Chennai - 600 001.

Seal

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INTRODUCTION

Preterm birth complicates 12% of all births. A birth is considered premature if the fetus is born before 37 weeks. Preterm labour and low birth weight represent major public health problems because of their associated mortality economic burden and long term disability [1].

The weight of the fetus at birth is the most important variable as far as the survival, growth and healthy development of a child is concerned. In cases of premature birth, the baby's vital organs are immature and incapable of readily adapting to the start of life outside the uterus.

The number of days spent in hospital and the costs of intensive neonatal therapy units are high. This cost to society goes beyond the financial cost because it permeates the psycho-emotional situation of the families involved [2].

Further there is also the possibility of future neuro-developmental abnormalities in the preterm baby [4, 5]. Although most preterm children are normal on neurological examination, the rates of neuromotor dysfunction are higher than in control groups. The spectrum of neurological deficits ranges from subtle degrees of neuromotor abnormality to cerebral palsy, with rates of cerebral palsy approaching 20% in the subset of infants with very low birth weight (where very low birth weight is defined as birth weight less

than 1,500 g) [5].

A higher prevalence of behavioural problems is reported in preterm children, including attention deficit hyperactivity disorder and formal conduct disorder [3]. Learning problems among low birth weight children have also been documented through teacher and parent ratings of school performance and direct assessments of academic skills in clinical settings; these children have been found to exhibit lower levels of achievement in reading, spelling and math [3].

Established risk factors for PLBW include [23, 41]

- Increasing maternal age (> 34 years)
- Teenage pregnancies
- Race-Preterm labour is more common among black women than whites.
- Height of mother less than 150cm
- Weight of mother less than 20 pounds
- Low socio-economic status
- Inadequate prenatal care,

- Drug/ alcohol/tobacco abuse
- Excessive physical activity
- Hypertension,
- Diabetes mellitus
- Multiple gestation/placental pathologies/fetal abnormalities
- Genitourinary tract infections.

Inspite of this it is estimated that 50% of premature births are idiopathic. This fact in itself justifies the need for further studies to identify the causes and risk factors that lead to onset of preterm labour [8, 9].

Evidence indicates that in some women maternal infection and inflammation of the lower and upper genital tract, as well as at sites distant from the pelvis play a major role in the etiology of preterm birth in some women [6, 7].

Periodontal disease is a chronic anaerobic inflammatory condition that affects many pregnant women. Emerging evidence supports a relationship between periodontal

disease and certain systemic conditions, including cardiovascular disease and diabetes mellitus [10].

The role of maternal periodontitis as a potential stressor having detrimental effects on pregnancy outcomes is a relatively new area of investigation.

Nevertheless, recent studies have demonstrated an association between maternal periodontal disease and multiple adverse pregnancy outcomes, including preterm birth, low birthweight, fetal growth restriction, and perinatal mortality [13].

The mechanisms by which periodontal disease and preterm birth are associated are not clear. It has been hypothesized that in the presence of severe periodontal disease, oral micro-organisms disseminate hematogenously to target the placenta, membranes, and fetus [14]. This bacterial challenge may result in increased cytokine expression and thereby precipitate preterm labor.

Given that many a pregnant women experience gingivitis—a preventable and treatable condition that could progress to periodontitis, a research to determine if oral diseases are independent risk factors for pre-term and low birth weight babies is clearly warranted.

C-reactive protein CRP is a marker for inflammation and infection. It is an acute phase protein, which is increased in severe periodontal diseases, and when maternal

CRP is increased it has been found to be associated with preterm deliveries [25].

Hence this study is being conducted to assess whether there is any risk of preterm labor with periodontal infection and increased C Reactive protein levels and also to assess the threat perception from such a relationship.

PERIODONTAL INFECTIONS

The periodontium is that part of the mouth that contains the gum, the crevices between the gums and the teeth, the surface roots, connective tissue and bone. Periodontitis is an inflammation of the periodontium, or one of the four tissues that support the teeth in the mouth [12]:

- the gingiva
- the cementum, or outer layer of the roots of teeth
- the alveolar bone, or the bony sockets into which the teeth are anchored
- the periodontal ligaments, which are the connective tissue fibres that connect the cementum and the gingiva to the alveolar bone.

Periodontitis is thought to occur in people who have preexisting gingivitis - an inflammation that is limited to the soft tissues surrounding the tooth and does not cause attachment or bone loss. Gingivitis is seldom painful and causes relatively minor signs, such as red, swollen and bleeding gums. But untreated gingivitis can progress to periodontitis.

Periodontitis is a chronic oral infection characterized by gram negative bacteria. The principal organisms implicated are

1. P.gingiivalis
2. F.nucleatum
3. E.corrodens
4. A.actinomycetumcomitants

Symptoms suggestive of Periodontitis

- Bleeding gums

- Suppuration of gums
- Halitosis and mobility of teeth

American Academy of Periodontology classification of the types of periodontal diseases [12]

Type I: Gingival Diseases: An inflammation or lesion of the gum characterized by changes of color, gingival form, position.

Type II: Chronic Periodontitis: An inflammation of the supporting structures of the teeth associated with plaque and calculus. It can be further classified as localized or generalized.

Type III: Aggressive Periodontitis: Characterized by a rapid rate of periodontal disease progression in an otherwise healthy individual in the absence of large accumulations of plaque and/or calculus. It can be further classified as localized or generalized.

Type IV: Periodontitis as a manifestation of systemic disease.

Type V: Necrotizing Periodontal Disease: Ulcerated and necrotic gums between the teeth and at the tooth Margins

Type VI: Abscess of the periodontium: A localized pus forming infection of the periodontal tissue.

Type VII: Periodontitis associated with endodontic lesions: localized deep periodontal pocket extending to the tip of the root of the tooth involving pulp death.

Type VIII: Developmental or acquired deformities and condition: Gingival disease or periodontitis started by localized tooth-related factors that modify or predispose to plaque accumulation.

Female Hormonal Alterations in pregnancy

Hormonal fluctuations in the female patient may alter the status of periodontal health [36]. Such changes may occur during puberty, the menstrual cycle, pregnancy, or menopause. Changes may also be associated with the use of oral contraceptives.

The most pronounced periodontal changes occur during pregnancy, as a significant proportion of pregnant women suffer from pregnancy gingivitis [36]. Estrogen, progesterone and chorionic gonadotropin (during pregnancy) all affect the microcirculatory system by producing the following changes [48]:

1. Swelling of endothelial cells and periocytes of the venules
2. Adherence of granulocytes and platelets to vessel walls
3. Formation of microthrombi
4. Disruption of the perivascular mast cells

5. Increased vascular permeability and vascular proliferation

The patterns of gingivitis appear to follow the normal cycle of hormonal changes and may be seen with varying degrees of significance. Of perhaps even greater importance than the above changes, however, is the shift in microbiota, which has been documented during these hormonal changes. **Kornman and Loeschein 1980** [49] found that during pregnancy, the ratio of bacterial anaerobes to aerobes and the proportions of *Porphyromonas gingivalis* increased.

Jensen and colleagues [50] studied the effect of hormone levels on the gingival status of 54 pregnant women, 23 non-pregnant women who received oral contraceptive therapy and 27 non-pregnant control subjects. They found that the pregnant women had a level of bacteroides species 55 times higher than that of the non-pregnant control group.

In addition, women who received oral contraceptive therapy had a 16-fold higher level of bacteroides species compared with women in the control group.

It appears, however, that the bacterial increases are cyclical in nature, because they follow the normal physiological changes and generally are of no consequence.

This pregnancy associated gingivitis is usually a transient and self-limiting condition. Gingival tissues return to their original healthy state postpartum when

estrogen and progesterone levels reach baseline values. Aside from these transient changes, pregnant women in good health are unlikely to experience any significant gingival response that would have serious clinical implications.

In some women especially in those who have a preexisting gingival pathology, this simple gingivitis can progress to periodontitis.

Hence all women with any kind of oral infection should seek treatment to prevent extension of the inflammatory process, and especially to avoid periodontal abscess formation and its resulting chronic bacterimia which could be a potential initiator of premature labour.

Role of Infection in Pre term Labour

Infection is considered one of the major causes of preterm labour responsible for somewhere between 30% and 50% of all cases [6, 7]. Bacterial infection of the chorioamnion or extraplacental membrane may lead to chorioamnionitis, a condition strongly associated with premature membrane rupture and preterm delivery.

The biological mechanisms involve bacteria induced activation of cell-mediated immunity, which leads to production of cytokines such as interleukins [IL-1 and IL-6], tumour necrosis factor alpha [TNF-α] and the ensuing synthesis and release of prostaglandins [26].

During normal pregnancy, the intra-amniotic levels of these mediators rise physiologically until a threshold level is reached, at which point labour, cervical dilatation and delivery are induced. Abnormal production of these mediators in the setting of infection triggers preterm labour and low birth weight [6].

However, many cases of histologically confirmed chorioamnionitis are not associated with active infection of the genitourinary tract and the results of culture are negative, both of which indicate that local infection is not the sole cause of this condition [46].

These findings led to speculation that an infection might be distant from the placental complex or the genitourinary tract and still present a risk for preterm labour, as a result of the indirect action of translocated bacterial products such as endotoxins (specifically lipopolysaccharides [LPS]) or the action of maternally produced inflammatory mediators (or both) [29].

Periodontal disease and maternal inflammatory response mechanism

The biological mechanism to support a link between maternal periodontal disease and preterm labour involves micro-organisms in the oral cavity that may enter the bloodstream passively through the inflamed periodontal pocket wall or through invasive oral procedures. The maternal periodontal infection then influences the fetoplacental unit in the following three ways [23, 26, 29].

1. Action of the proinflammatory mediators

Periodontal diseases are associated with chronic gram negative anaerobic infections resulting in local and systemic elevations of proinflammatory prostaglandins, including prostaglandin E2 (PGE2) and cytokines (IL-1, IL-6, and TNF-α). It is the artificially high levels of the prostaglandins that foster premature labour.

2. The action of the periodontal reservoir of bacterial lipopolysaccharides (LPS)

The oral micro-organisms themselves are not directly implicated in the preterm labour.; rather, bacterial lippolysaccharide(LPS) (endotoxin) stimulation occurs in response to localized non-disseminating substantaneous infection with porphyromonas gingivalis (a common periodontal pathogen).

It is said that these bacterial lipopolysaccharide (LPS) can mediate the release of

fever inducing IL-1 (interleukin-1), TNFα (tumor necrotic factor α) and the hepatic release of acute phase proteins such as C-reactive protein (CRP). LPS can also target the placenta to induce the placental production of IL-1 and IL-6 resulting in inflammation of the placenta without reaching the fetal circulation [23, 26, 29].

Production of IL-1, IL-6. TNFα, CRP and secondarily PGE2 (prostaglandin E2) induces uterine contractions and modulates placental blood flow and could also result in preterm parturition [26].

3. Direct assault of micro-organism on the fetoplacental unit

This mechanism involves translocation of periodontal pathogens to the fetoplacental unit, through the blood.

Thus periodontitis leads to a cascade of events that involve systemic maternal inflammatory responses, as well as inflammation of the fetal-placental unit resulting in abnormal pregnancy outcomes.

Periodontal Disease and blood CRP levels

Periodontal disease being chronic and cyclic in nature provides an opportunity for repeated hematogenous dissemination of periodontal pathogens and direct microbial exposure of the vasculature, the liver and the placental-fetal unit among pregnant

women.

The organisms can easily ingress into the peripheral circulation by way of regular chewing or any dental manipulations such as brushing or flossing. The severity of the recurrent bacteremia is said to be the principal cause of the observed association between periodontal disease and increased serum CRP levels.

Tillete and Francis in 1930 [27] were the first to discover the C Reactive protein an acute phase protein which is the primary response of the body to injury.

The C Reactice protein is so named because it forms a precipitate in a non-specific somatic reaction with the pneumococcal capsular polysaccharide in the presence of calcium ions.

In the body CRP can bind to a wide variety of substances derived from both damaged autologous cells and from micro-organisms. Complexed CRP can activate the compliment system and thereby serves as a protective mechanism to the body.

Clinical measurement of CRP is valuable as a screening test for organic diseases and as an index for disease activity and response to therapy in certain inflammatory and ischemic conditions.

C-reactive protein (CRP) is synthesized by the liver in response to the

inflammatory cytokines interleukin IL-6, IL-1, and TNFα tumor necrosis factor-alpha [29]. Circulating CRP levels are a marker of systemic inflammation and are associated with periodontal disease [30], which causes elevation of proinflammatory cytokines and prostaglandin. Interestingly, standard non-surgical periodontal therapy has been found to cause a decrease in serum CRP levels [31].

Evidence supporting the association between periodontitis and CRP is based mainly on studies in men and non-pregnant women. There are very few studies of periodontitis and CRP in pregnant women. This is an important area for study because systemic inflammation plays a major role in the pathogenesis of preterm delivery.

CRP has been associated with adverse pregnancy outcomes, including preeclampsia, intrauterine growth restriction, and preterm delivery. Chronic infections like intrauterine infection and chorioamnionitis are linked to both preterm birth and elevated CRP levels. Furthermore, periodontal disease has been associated with increased risk of preterm low birth weight, low birth weight, and preterm birth [32, 42].

Therefore, CRP might be a plausible mediator of the association between periodontitis and adverse pregnancy outcomes [28].

Treatment of Periodontitis

The therapeutic goals of periodontal therapy are [35]

1. To alter or eliminate the microbial etiology and contributing risk factors for periodontitis, thereby arresting the progression of disease and preserving the dentition in a state of health, comfort, and function with appropriate aesthetics

2. To prevent the recurrence of periodontitis

3. Regeneration of the periodontal attachment apparatus, where indicated, may be attempted.

In general the treatment includes [35]

- Maintenance of meticulous oral hygiene by daily brushing and flossing.
- Plaque control by mechanical therapy, i.e., supra- and sub-gingival scaling and root planing to remove microbial plaque and calculus along with adjunctive microbial therapy.
- If this initial therapy resolves the periodontal condition, supportive periodontal therapy is scheduled at appropriate intervals.
- If the periodontal condition is not resolved, periodontal surgery should be considered to as a last resort to correct anatomic defects, and/or to regenerate hard and soft tissues.

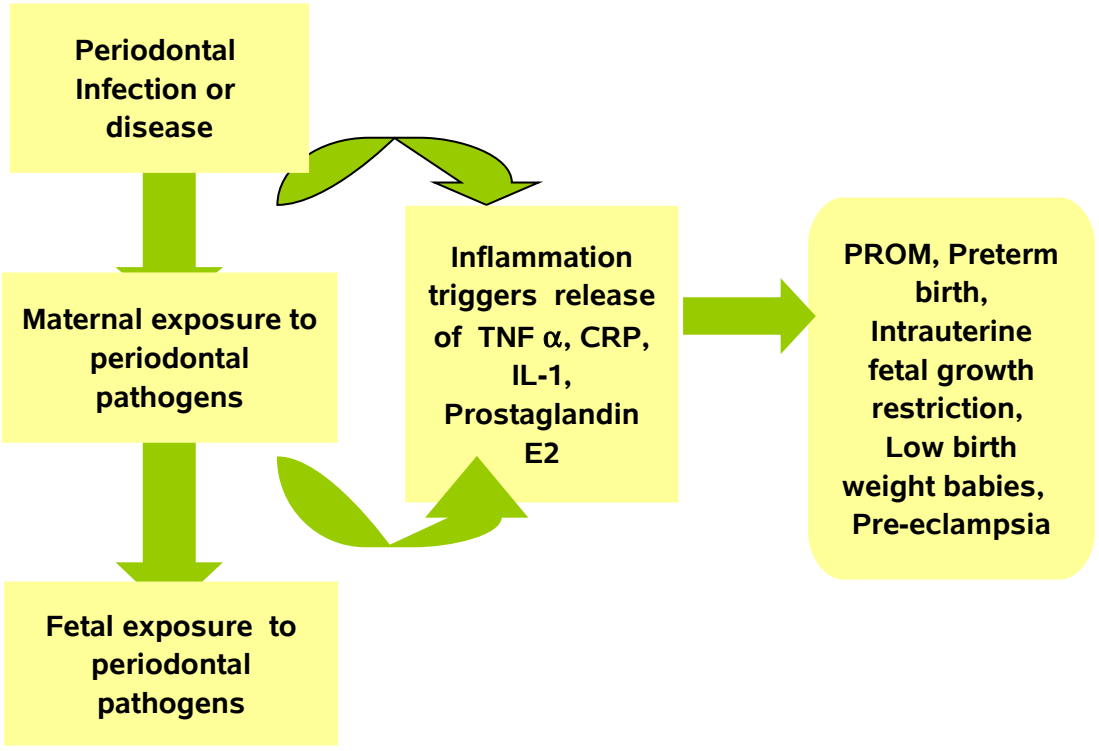
Preterm Neonate

Healthy Periodontium

Diseased Periodontium

Diseased Periodontium

Proposed Model for Relationship between Periodontitis and Pre-term labour



AIM OF STUDY

- To study the relationship between periodontitis and preterm labour.
- To investigate whether C Reactive Protein is the possible mediator of the association between periodontitis and preterm labour.

LITERATURE REVIEW

Miller in 1891 [11] published the theory of "focal infection". On the basis of this theory, oral foci of infection were considered responsible for a number of regional and systemic diseases, such as tonsillitis, pneumonia, endocarditis and septicemia. However, the lack of scientific evidence condemned this theory to dormancy.

It was 100 years later, in the early 1990s that **Collins and colleagues** [23]

hypothesized that oral infection, such as periodontitis, could act as a source of bacteria and inflammatory mediators that could disseminate systemically to the feto-placental unit, via the blood circulation, and induce pregnancy complications.

In a series of landmark animal studies in which pregnant hamsters were injected with the periodontal pathogen *Porphyromonas gingivalis*, Collins and colleagues[24] found that infection led to smaller fetuses (approximately 20 percent reduction in weight) and to an increase of inflammatory mediators (TNF α and PGE2) at the site of infection and in the amniotic fluid.

In subsequent experiments, in which periodontal disease was induced in pregnant hamsters, the investigators found similar results in terms of fetal growth. These were the first proof-of-principle experiments to suggest a possible association of periodontal disease with adverse pregnancy outcomes.

Four separate lines of evidence currently relate oral infection to pregnancy outcome: microbiological studies, case-control studies, prospective longitudinal studies, and intervention studies.

The first line of evidence to support a relationship between periodontitis and preterm labour is found in several microbiological studies of amniotic fluid, maternal and fetal cord serum, and gingival crevicular fluid (GCF).

Damare et al (1997) [19] conducted a small study showing a high correlation

between in the levels of PGE2 and IL-1ß in maternal gingival crevicular fluid and amniotic fluid and the occurrence of premature labour.

Offenbacher et al (1998) [14] conducted a similar study and found that the gingival crevicular fluid GCF of 48 mothers of pre-term low birth weight infants had higher levels of PGE2 than mothers of infants with normal birth weight.

Offenbacher et al (1999) [15] analyzed blood samples from fetal cords for the presence of immunoglobulin M (IgM) antibody against various periodontal pathogens. Of the serum samples obtained from preterm low birth weight infants, 33.3% tested positive for IgM against the test micro-organism. However, only 17.9% of the normal birth weight samples tested positive.

Each of the above studies suggest that periodontal infection is a source of microbial products causing an inflammatory response that affects the pregnancy outcome. The studies also suggest that maternal periodontal infections, resulting in blood-borne micro-organisms that can translocate to the fetus, provide a systemic challenge to the fetus and induce an immunologic response.

The second line of evidence comes from examining case-control studies.

Offenbacher et al (1996) [13] studied 124 pregnant or postpartum mothers and found that periodontal disease is a statistically significant risk factor for preterm labour . There was an odds ratio (OR) of 7.9 (95% CI, 6.27–9.58) for preterm cases (n = 93). In

other words, mothers with periodontal infection had more than seven times the risk of delivering a pre-term low birth weight infant.

Dasanayke et al (1998) [20] reported the findings of a one-to-one matched case-control study (N=55 pairs) that evaluated whether the poor oral health of a pregnant woman at the time of delivery has an effect on the birth weight of the infant. Analysis indicated that mothers with healthier surface areas of gingival tissue had a lower risk of giving birth to a low birth weight infant (OR=0.3, 95% CI=0.12-0.72). Therefore, they concluded that poor periodontal health of the mothers is a potential independent risk factor for the delivery of low birth weight babies.

The third line of evidence comes from two prospective studies. **Jeffcoat et al (2001)** [21] conducted a large prospective study with approximately 1,300 mothers and concluded that maternal periodontitis is an independent risk factor for preterm birth. Periodontitis in the mother was assessed at 21 to 24 weeks gestation. The odds ratio for severe periodontitis was reported at 4.45 (95% CI, 2.16–9.18) for infants born before 37 weeks gestational age. The odds ratio increased to 7.07 (95% CI, 1.70–27.40) for delivery before 32 weeks.

Offenbacher et al (2001) [13] report on 814 deliveries with periodontal examinations performed at enrolment and again within 48 hours postpartum, with blinded examiners. The results provide strong support for the hypothesis that maternal periodontal disease is a risk factor for preterm birth.

Madianos et al (2001) [18] reported that progression or worsening of periodontal disease is associated with a significant risk of preterm labor as compared with a group of pregnant women with gum disease that did not worsen throughout their pregnancy. This was one of the first studies to evaluate the effects of periodontal disease progression and birth outcomes. All of the women had oral examinations before 26 weeks gestation and again within 48 hours of delivery. The study showed that thirty-two women (36%) had periodontal disease that progressed during pregnancy with a corresponding rate of preterm birth of 47%, compared with 27% among those with stable oral disease. After controlling for confounding factors, women with progressive disease were nearly five times more likely to give birth before 37 weeks, compared with those whose disease did not progress.

The fourth line of evidence comes from three intervention studies using periodontal treatment for pregnant mothers with existing oral health problems.

Mitchell-Lewis et al in (2001) [22] showed a prevalence of 18.9% preterm low birth weight infants without periodontal treatment for the pregnant mothers versus 13.5% with treatment. Periodontal treatment included oral hygiene instruction and full-mouth debridement, including scaling, polishing with fluoridated paste, and dental sealants.

Lopez et al. (2002) [16] conducted a randomized controlled trial with 351 women with periodontal disease. Results suggest that periodontal disease is an independent risk

factor for preterm low birth weight. Periodontal therapy consisted of plaque control instructions, scaling, root planing, and chlorhexidine rinse once a day. In addition, 18% of the treatment group had severe periodontitis and were given metronidazole 250 mg plus amoxicillin 500 mg three times a day for seven days.

The incidence of preterm labour in the group, which was treated, was 1.84% (3/163)—significantly lower than in the control group, which was10.11% (19/188). Women with periodontal disease have more than four times greater risk of having a preterm infant than periodontally healthy women. Mothers with periodontal disease had an odds ratio of 4.70; 95% confidence interval, 1.29 to 17.13.

Jeffcoat et al. (2003) [17] also conducted a randomized controlled trial with 366 women with periodontitis between 21 and 25 weeks gestation to determine whether treatment of periodontitis reduces the risk of spontaneous preterm birth. Subjects were randomly assigned to one of three treatment groups:

(1) Dental prophylaxis (tooth cleaning and polish) plus placebo capsule three times a day

(2) Scaling and root planing plus placebo capsule three times a day; and

(3) Scaling and root planing plus metronidazole 250 mg three times a day for one week

Results showed that the rate of preterm birth at <35 weeks was 4.9% for the first group (prophylaxis plus placebo), 3.3% for the third group (scaling and root planing plus metronidazole), and the lowest rate of 0.8% for the second group (scaling and root planing plus placebo). The rate of preterm birth was 6.3% in the reference or untreated group. Results also show as much as an 84% reduction in spontaneous preterm birth at <35 weeks gestation in subjects receiving scaling and root planing compared with the prophylaxis plus placebo group.

Periodontitis and C Reactive Protein

Tilette and France in 1930[27] were the first persons to discover the C Reactive protein which is an acute phase protein produced by the body in response to injury.

M.B. Pepys in 1981 [27] proposed that an elevated serum concentration of CRP is an evidence of active tissue damaging processes and measurement of CRP levels forms a screening test for organic diseases and inflammatory states. Increased CRP levels were also a very early and sensitive response to different forms of microbial infections.

Ebersole et al in 1997 [44] compared the levels of both CRP and haptoglobulin in the serum of adult with periodontitis and in normal subjects using ELISA and found that both were elevated in the sera of adults with periodontitis. Statistically significant decrease in the values occurred after the treatment of periodontitis.

Novac et al in 2001 [45] examined the levels of CRP in patients with periodontitis and correlated it to the severity of periodontal disease and to periodontal microflora. Results obtained showed that the extent of increase in CRP levels in patients depends on the severity of the disease after adjusting for age, body mass index and smoking.

The subgingival plaque samples were then examined for the presence of bacteria known to cause periodontitis by immunoflourescence microscopy. Presence of periodontal pathogens was positively associated with elevated CRP levels.

Various studies have shown that increased CRP levels are associated with preterm labour and low birth weight babies.

Dr. Waranuch Pitiphat et al (2005) [52] in a study on singleton pregnancies, found that very high levels of maternal plasma CRP in early pregnancy were associated with increased risk of preterm delivery. Compared with women with normal CRP levels, those with elevated CRP levels (≥ 8 mg/liter) had a greater than twofold higher odds of preterm delivery. The association was stronger for cases who experienced spontaneous preterm delivery versus induced preterm delivery.

In 2006 Waranuch Pitiphat et al [28] conducted a cohort study, where they measured plasma CRP in 35 subjects with periodontitis (i.e., at least one site with ≥3 mm of alveolar bone loss) and a random sample of 66 periodontally healthy subjects matched on age and race/ethnicity. The mean CRP level

was 65% higher (95% confidence interval: −2%, 180%; P = 0.06) in women with periodontitis

(2.46 ± 0.52 mg/l) than in controls (1.49 ± 0.22 mg/l), after adjusting for factors related to CRP levels, including age, race/ethnicity, pre-pregnancy body mass index, alcohol intake, education, income, and gestational age at blood collection.

Recently **Amanda et al in 2008** [51] conducted a study in African American pregnant women and concluded that moderate/severe periodontal diseases were significantly associated with elevated CRP levels (adjusted OR: 4.0; 95% confidence interval [CI]: 1.2 to 8.5).

All the above studies suggest that periodontitis may increase CRP levels in pregnancy. CRP could potentially mediate the association of periodontitis with adverse pregnancy outcomes.

MATERIALS AND METHODS

Study Population

The study is a retrospective case–control study conducted amongst the post-natal

women who delivered at the ISO and Govt KG Hospital, Chennai during the period from April 2007 to April 2008. The study received ethical clearance from the board.

The hospital birth register was scrutinized each day to identify all cases of preterm delivery, defined as those mothers who delivered an infant before 37 weeks gestation. The control (mothers who delivered an infant after 37 weeks gestation) was selected daily from the birth register at the same time as the cases.

A written consent of the women was obtained before their enrolment in the study and all data was collected within 72 h of delivery. During the period of study a total of 200 post partum mothers were included for the study 100 cases(Pre-term deliveries) and 100 controls (term deliveries). The case-control ratio was 1:1.

Inclusion Criteria

- Cases were Primiparous /Multiparous women who delivered a single live infant before 37 weeks of gestation and after achieving viability.
- Controls were either primiparous / multiparous women who delivered a single live infant at 37 completed weeks of gestation.
- Women who have had no medical/surgical illness which could have been a predisposing factor for preterm labour.
- No evidence of infection elsewhere at present.

Exclusion Criteria

- Women with genito-urinary tract infections
- Medical /surgical illness which might predispose to preterm labour
- Women who had therapeutic induction of labour
- Women who delivered multiple fetuses /still born infants
- Women who had only a localized form of periodontitis

A detailed history was elicited from the patients regarding the presence of any symptoms pertaining to dental infections, history of abnormal vaginal discharge, and

history regarding their previous deliveries. Additional information was also obtained from the maternal case sheet.

Assessment of Gestational age at delivery

Gestational age was calculated from the last menstrual period (LMP) if the patient was sure of her dates. In case of any discrepancy in the calculated dates or when the LMP was not known a first trimester ultra sound was used wherever available to calculate the gestational age. These findings were confirmed by a post-natal examination of the baby by the pediatrician to assess and confirm the maturity.

A detailed general examination and a meticulous local examination were performed.

The patients were subjected to a speculum examination to note the presence of any abnormal discharge. A drop of Potassium Hydroxide was added to the secretion and the development of a fishy odour was taken as a positive test for Bacterial Vaginosis. These patients were excluded from the study.

Urine was collected in a sterile container and sent to the microbiology lab where culture was done. Those with urinary tract infection were also excluded from the study.

Periodontal Examination

Study participants underwent an intra-oral examination by a qualified

periodontologist within 72 hours postpartum. The examination was carried out using a mouth mirror and William's graduated periodontal probe.

Periodontal Evaluation

The probing depth and the clinical attachment loss (CAL) were the parameters that were measured.

Probing Depth

The distance in mm from the cemento-enamel junction to pocket base was defined as probing depth. At least 6 areas of each tooth (buccal-mesial, mid-buccal, buccal-distal, lingual-mesial, mid-lingual and lingual-distal) was examined using the periodontal probe. The highest score in mm for any tooth was recorded for probing depth.

Clinical Attachment Loss (CAL)

Attachment loss is a more accurate measure of disease severity than probing depth and is defined as the distance in mm between the cemento-enamel junction and the base of the periodontal pocket. CAL was first calculated for each tooth separately. The mean CAL value was then computed.

Periodontitis was classified as localized or generalized depending on whether < 4 or ≥ 4 sites showed a probing depth of > 3 mm and CAL >1 mm. Subjects with

generalized periodontitis alone were included for the study.

Severity of periodontitis was classified as follows (AAP 1999):

- Periodontal Health was defined as CAL< 1 mm
- Mild Periodontitis-CAL in the range of 1.1 -2.9 mm
- Moderate Periodontitis-CAL in the range of 3-5 mm
- Severe Periodontitis-CAL >5 mm

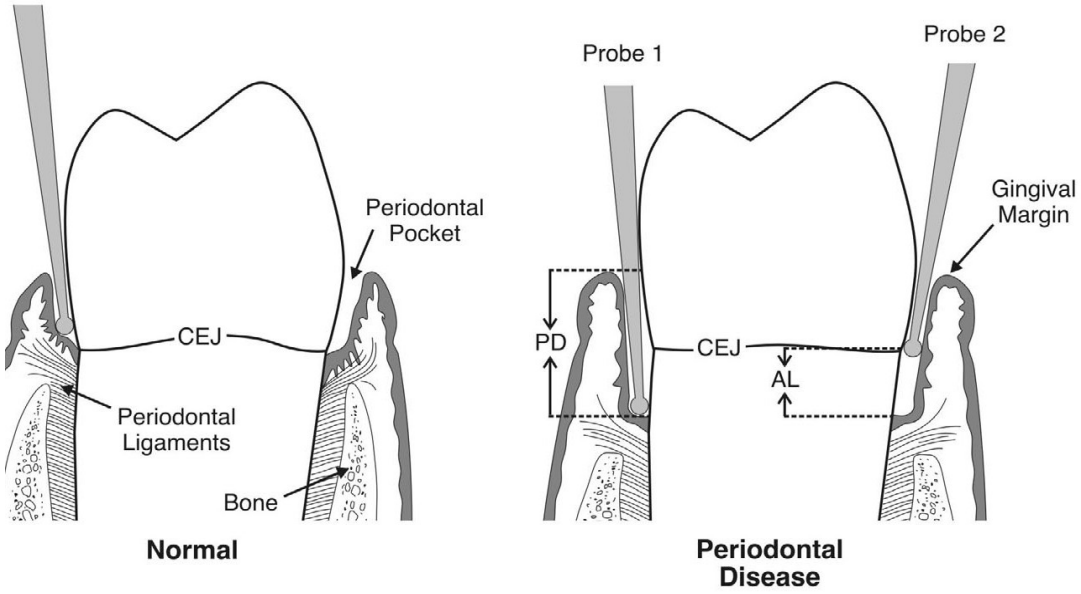
Biochemical Investigations

5 ml of blood was drawn from every subject for routine investigations and also for estimation of C - Reactive protein (CRP) levels.

Mouth mirror and William's graduated periodontal probe

Determination of probing depth using Williams Periodontal probe

Measurement of probing depth and clinical attachment level



Probe 1 (left) is measuring probing depth, the distance from the gingival margin to the base of the periodontal probe, probe2 (right) is measuring the distance from the gingival margin to Cementum-the Cemento-Enamel Junction (CEJ).Attachment Loss=probe1-probe2

Kit for CRP estimation

Instrument used for CRP estimation

Estimation of C - Reactive Protein by Nephelometry

Principle

Polystyrene particles coated with monoclonal antibodies specific to human CRP are aggregated when mixed with samples containing CRP. These aggregates scatter a beam of light passed though the sample. The intensity of the scattered light is proportional to the concentration of the relevant protein in the sample. The result is evaluated by the comparison with a standard of known concentration. The assigned value of CRP in CN Rheumatology standard SL is standardized against the interaction reference preparation BCR-CRM 470.

Composition

Cardiophase hsCRP reagent consists of a suspension of polystyrene particles coated with monoclonal antibodies to CRP.

Armamentarium

1. BN System
2. Cardiophase hsCRP reagent
3. Five vials containing 5ml each of three vials containing each.
4. N Rheumatology standard SL
5. N/T Rheumatology controls SL/1 and SL/2
6. Apolipoprotein

7. N supplementary reagent/precipitation

8. N Diluent

Procedure

1. Allow reagents and samples to equilibrate to room temperature before use on the instrument.

2. On the instrument, samples should run at approximately the same ambient temperature (maximum 3° deviation) as the measurements used for recording the reference curve.

Assay Protocols

The assay protocols, for serum as well as plasma, are given in the instruction manual and software of the instrument. All steps are performed automatically by the system.

Establishment of Reference curves

Reference curves are generated by multi-point calibration. Serial dilutions of N Rheumatology standard SL are automatically prepared by the instrument using N Diluent. The standard dilutions are to be used within four hours. The reference curve is valid for four weeks and can be used beyond this period of time, as long as controls with

corresponding method depending target values, e.g., N/T Rheumatology Controls SL/1 and SL/2 or Apolipoprotein Control Serum CHD, are reproduced within their respective confidence interval. If a different lot of reagent is used, a new reference curve must be generated. The exact measuring range depends upon the concentration of the protein in which lot of N Rheumatology Standard SL.

Assay of specimens

Samples are automatically diluted 1:400 (CRP1) or 1:30 in the cardiophase hsCRP assay protocol (CRP2) with N Diluent. The diulent samples must be used within four hours. If the results obtained are outside the measuring range, the assay can be repeated using a higher or lower (only in the CRP1 assay protocol) dilution of the sample.

Results

The results are evaluated automatically by the analyzer and are represented in mg/L or in a unit selected by the instrument.

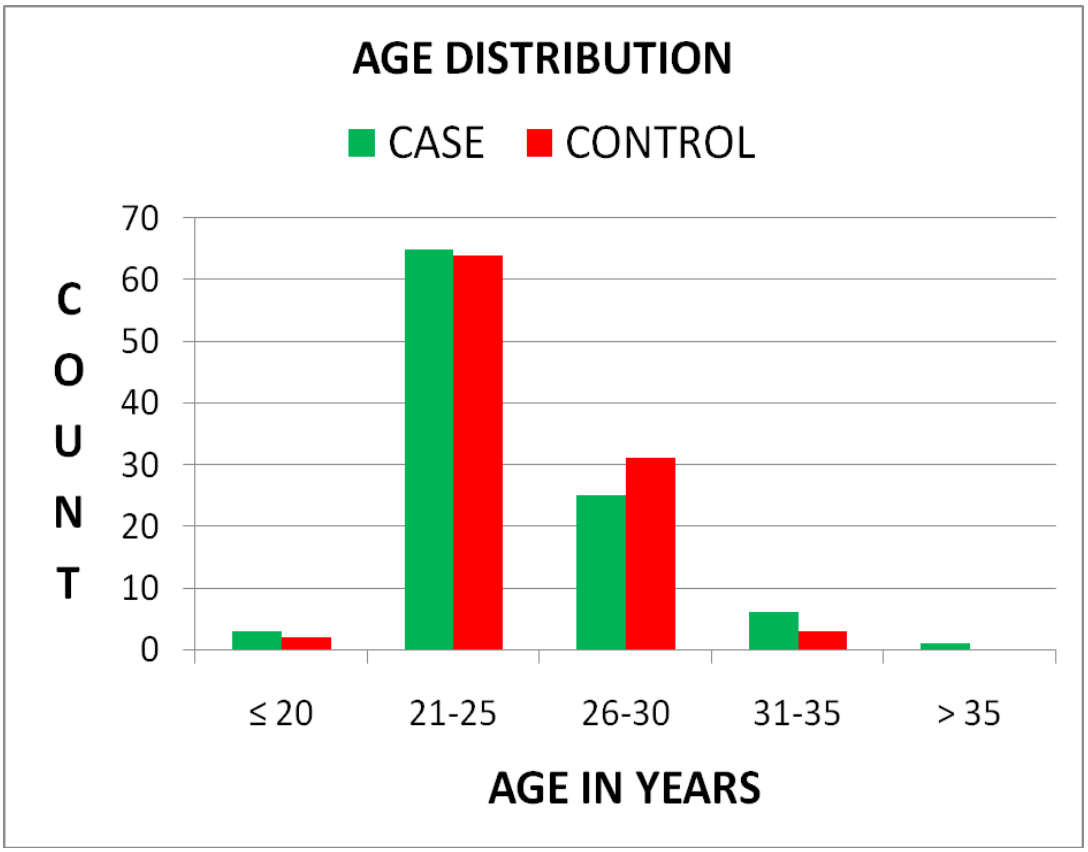
RESULTS

Age distribution (Table1)

Sr No	Age in Years	Case		Control	
		Number	Percentage	Number	Percentage
1	≤ 20	3	3%	2	2%
2	21-25	65	65%	64	64%
3	26-30	25	25%	31	31%
4	31-35	6	6%	3	3%
5	> 35	1	1%	-	-

p value >0.05 → Not Significant

The age distribution among the case and control groups was similar. The average child bearing age in both groups was about 24 years.

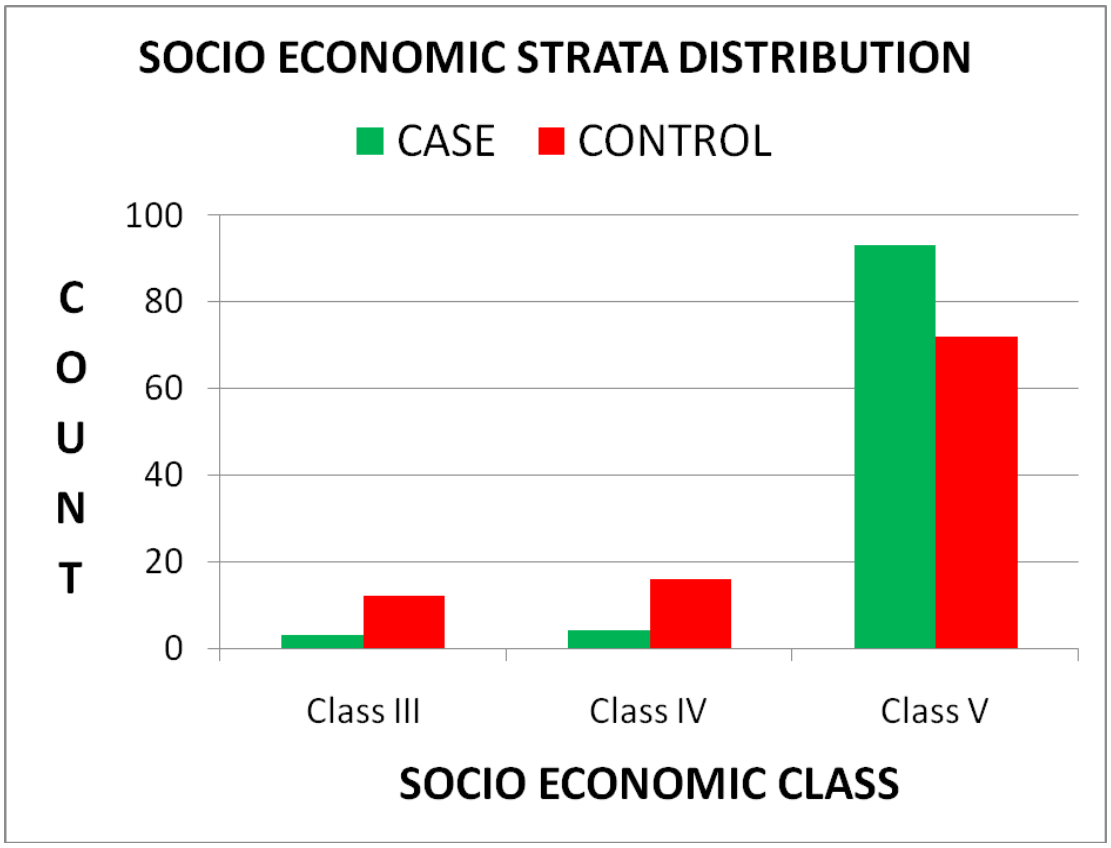

AGE DISTRIBUTION
CASE
CONTROL
COUNT
70
60
50
40
30
20
10
0
≤ 20
21-25
26-30
31-35
> 35
AGE IN YEARS

Socio-Economic strata distribution (Table2)

Sr No	Socio-Economic Group	Case		Control	
		Number	Percentage	Number	Percentage
1	Class III	3	3%	12	12%
2	Class IV	4	4%	16	16%
3	Class V	93	93%	72	72%

p value $<0.05 \rightarrow$ Significant

Cases were from a comparatively lower socio-economic stratum when compared to controls.


SOCIO ECONOMIC STRATA DISTRIBUTION
CASE
CONTROL
COUNT
100
80
60
40
20
0
Class III
Class IV
Class V
SOCIO ECONOMIC CLASS

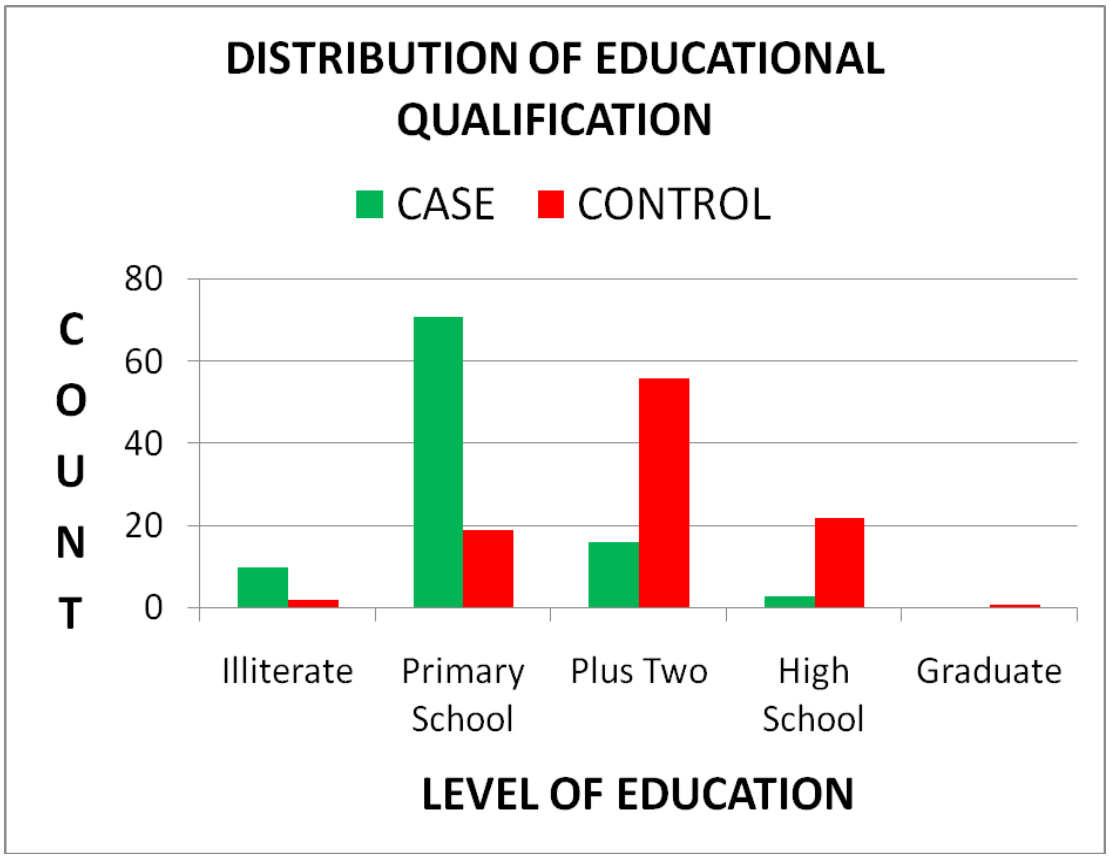
Educational Qualification

(Table3)

Sr No	Level of Education	Case		Control	
		Number	Percent-age	Number	Percent-age
1	Illiterate	10	10%	2	2%
2	Primary School	71	71%	19	19%
3	Plus Two	16	16%	56	56%
4	High school	3	3%	22	22%
5	Graduate	-	-	1	1%

p value<0.05→ significant

Cases had a relatively lesser educational background than the controls.


DISTRIBUTION OF EDUCATIONAL QUALIFICATION
CASE
CONTROL
COUNT
80
60
40
20
0
Illiterate
Primary School
Plus Two
High School
Graduate
LEVEL OF EDUCATION

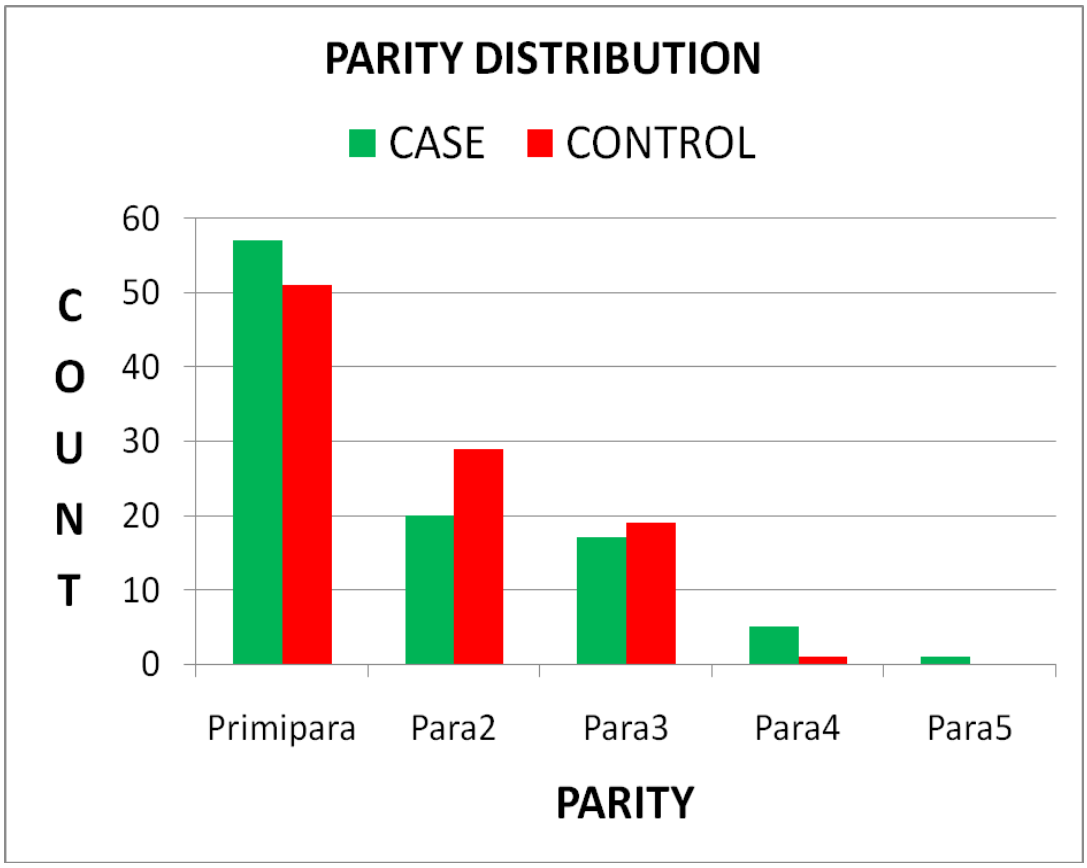
Parity Distribution

(Table4)

Sr No	Parity	Case		Control	
		Number	Percent-age	Number	Percent-age
1	Primipara	57	10%	51	51%
2	Para2	20	71%	29	29%
3	Para3	17	16%	19	19%
4	Para4	5	3%	1	1%
5	Para5	1	-	-	-

p value > 0.05 → Not Significant

Primiparas were the largest in number both amongst the cases and controls. Similarly all other parity groups seemed to be equally distributed among both the groups.


PARITY DISTRIBUTION
CASE
CONTROL
COUNT
60
50
40
30
20
10
0
Primipara
Para2
Para3
Para4
Para5
PARITY

Previous History of Pre-term Births (Table5)

Study Population	Previous History of Pre-term Births			
	Yes		No	
	Number	Percent-age	Number	Percent-age
Cases (n=43)	8	18.6%	35	81.39%
Controls (n=48)	2	< 1%	46	95%

p value < 0.05→ significant

Previous preterm labour was more common amongst the multipara in the case group (18.6%) than in the control group (< 1%).

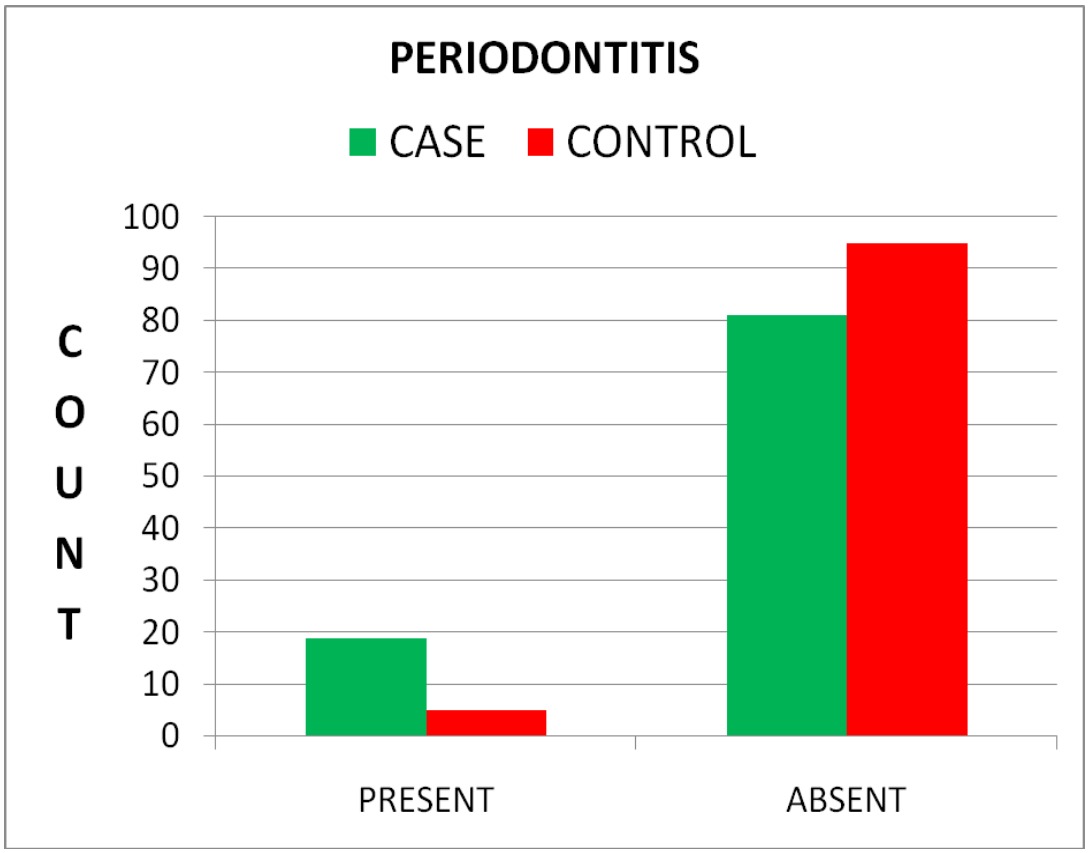
Periodontitis

(Table 6)

Periodontitis	Cases		Controls	
	Number	Percentage	Number	Percentage
Present	19	19%	5	5%
Absent	81	81%	95	95%

p value < 0.05→Significant

Periodontitis was seen more commonly among cases than controls.

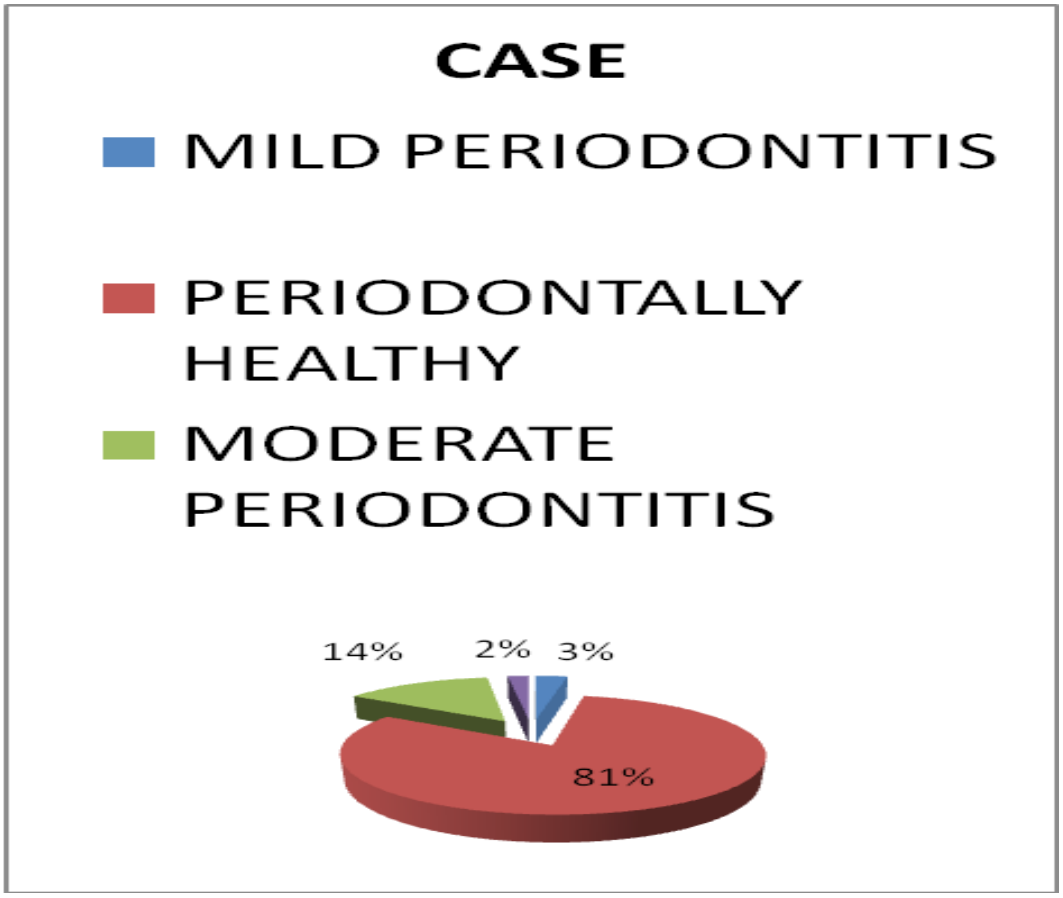

PERIODONTITIS
CASE
CONTROL
COUNT
100
90
80
70
60
50
40
30
20
10
0
PRESENT
ABSENT

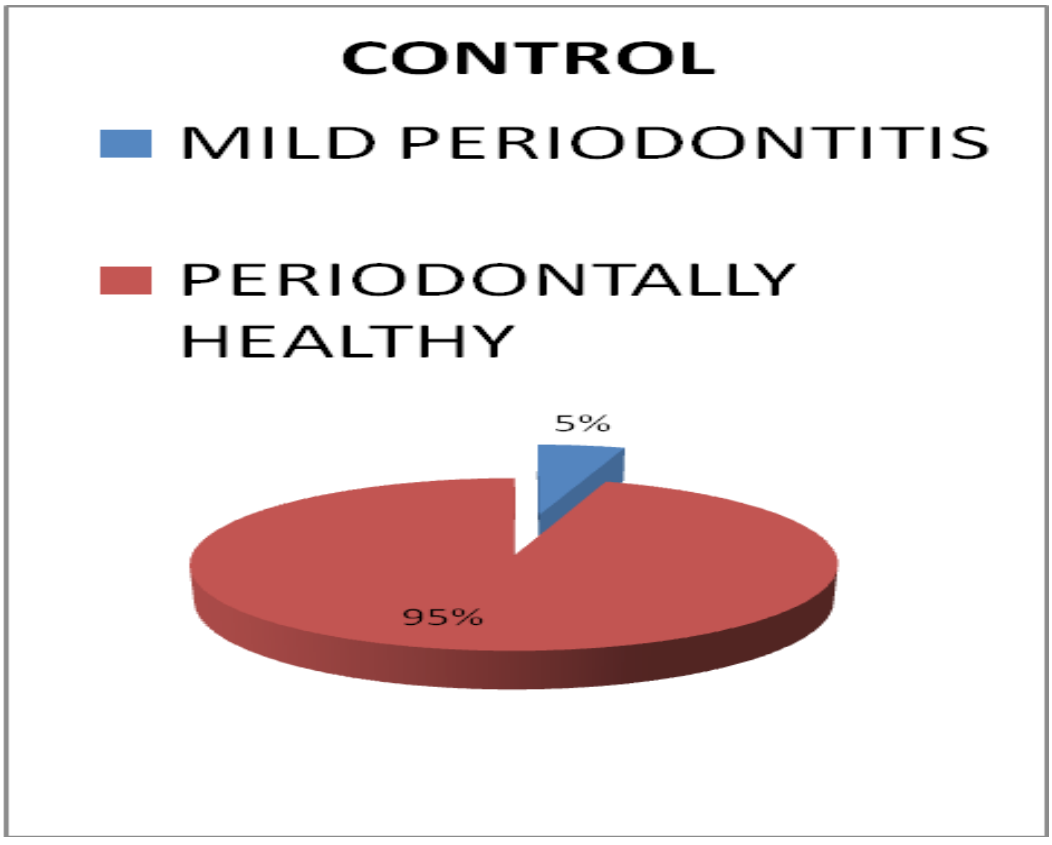
Degree of Periodontitis

(Table 7)

Degree of Periodontitis	Case		Control	
	Number	Percent-age	Number	Percent-age
Mild	3	3%	5	5%
Moderate	14	14%	-	-
Severe	2	2%	-	-

All 5 cases of periodontitis in the control population belonged to the category of mild periodontitis. Amongst the cases 3 had mild periodontitis, 14 had moderate periodontitis and 2 had a severe degree of periodontitis.


CASE
MILD PERIODONTITIS
PERIODONTALLY HEALTHY
MODERATE PERIODONTITIS
14%
2%
3%
81%


CONTROL
MILD PERIODONTITIS
PERIODONTALLY HEALTHY
5%
95%

C Reactive Protein (CRP) Levels (Table8)

Sr No	CRP level in mg/l	Study Population			
		Case		Control	
		Number	Percentage	Number	Percentage
1	<1	11	11%	36	36%
2	1-2	69	69%	63	63%
3	>2	20	205	1	1%

p value<0.05→significant

Levels of CRP was comparatively more among cases than controls.

Comparison of CRP levels in mothers with periodontitis in the case group (Table 9)

Degree of Periodontitis	Mean CRP level in mg/l
Mild	2.13
Moderate	2.75
Severe	4.05

The CRP levels were seen to increase with increasing severity of periodontitis. The level was highest in those women with severe periodontitis. On applying the anova and post-hoc tests this was found to be statistically significant.

Gestational age at delivery

(Table 10)

Study Population	Mean gestational age at delivery in weeks

Case	33.9
Control	39.1

The mean gestational age at delivery was 33.9 weeks in the case group and it was 39.1 weeks in the control group.

Comparison of the gestational age at delivery in women with periodontitis in the case group (Table11)

Degree of Periodontitis	**Mean gestational age at delivery in weeks**

Mild (n=3)	35.6
Moderate (n=14)	33.3
Severe (n=2)	30.5

Within the case group the gestational age at delivery was lesser in those with severe periodontitis when compared with mild and moderate varieties of periodontitis. On applying the anova and post-hoc tests this was found to be statistical significant.

Correlation between degree of Periodontitis, gestational age at delivery and CRP levels

(Table12)

Degree of Periodontitis	**Mean gestational age at delivery in weeks**	**Mean CRP Levels in mg/l**
Mild	35.6	2.13
Moderate	33.3	2.75
Severe	30.5	4.05

With increase in the severity of periodontitis there was a corresponding increase in CRP level and a decrease in the gestational age at delivery.

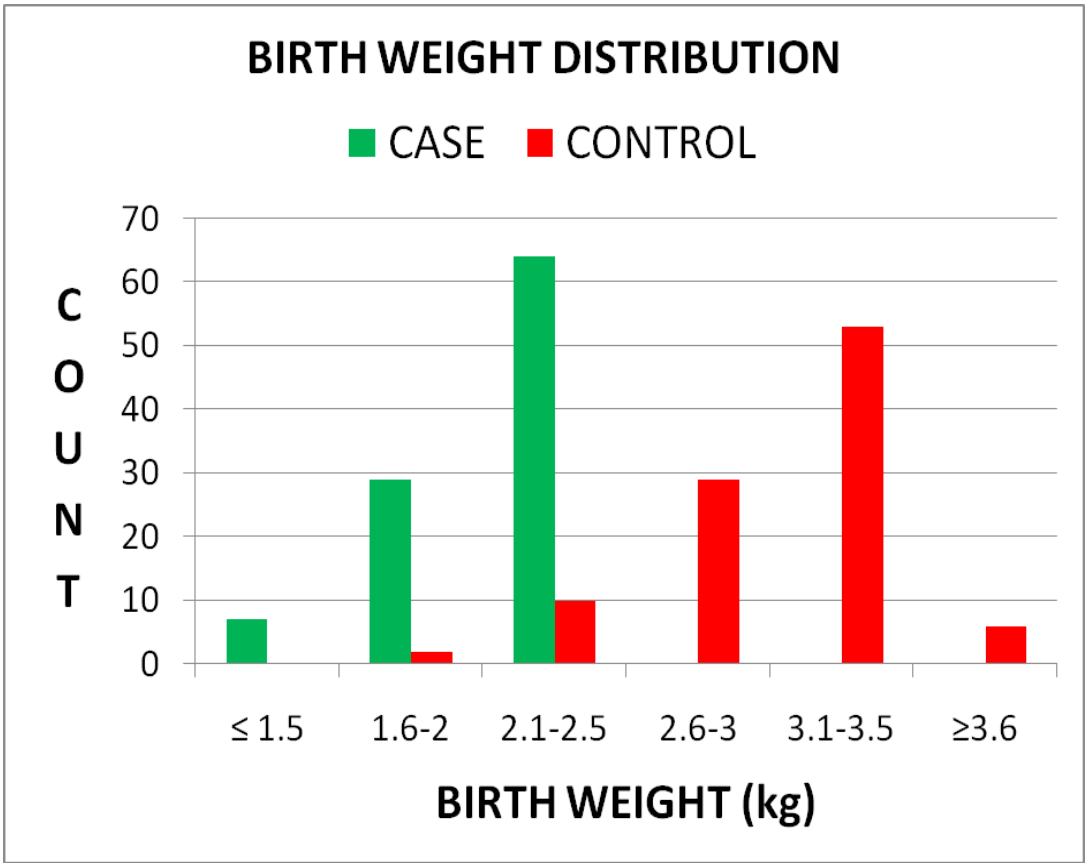
Birth Weight Distribution

(Table13)

Sr No	Birth weight (kg)	Case		Control	
		Number	Percentage	Number	Percentage
1	≤1.5	7	7%	-	-
2	1.6-2	29	29%	2	2%
3	2.1-2.5	64	64%	10	10%
4	2.6-3	-	-	29	29%
5	3.1-3.5	-	-	53	53%
6	≥3.6	-	-	6	6%

P value < 0.05 → significant.

The babies of the mothers in the case group weighed relatively less than that in the controls. Whereas the average birth weight among the cases was 2.07 kg whereas those among controls was 3.04 kg. There were only two babies with birth weight < 2 kg in the control group.



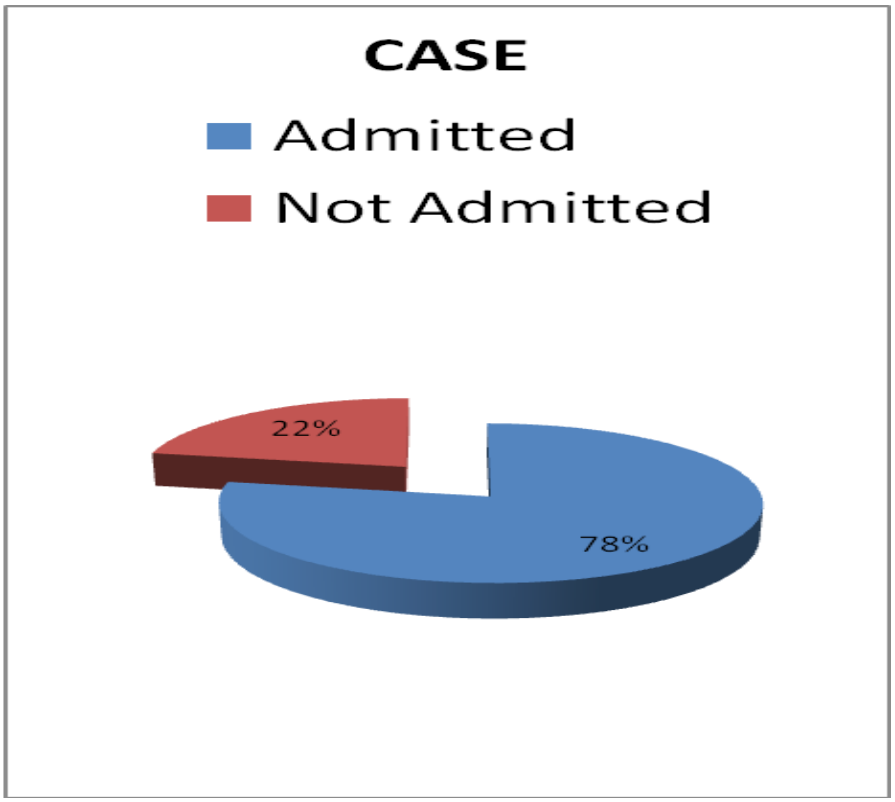
NICU Admissions

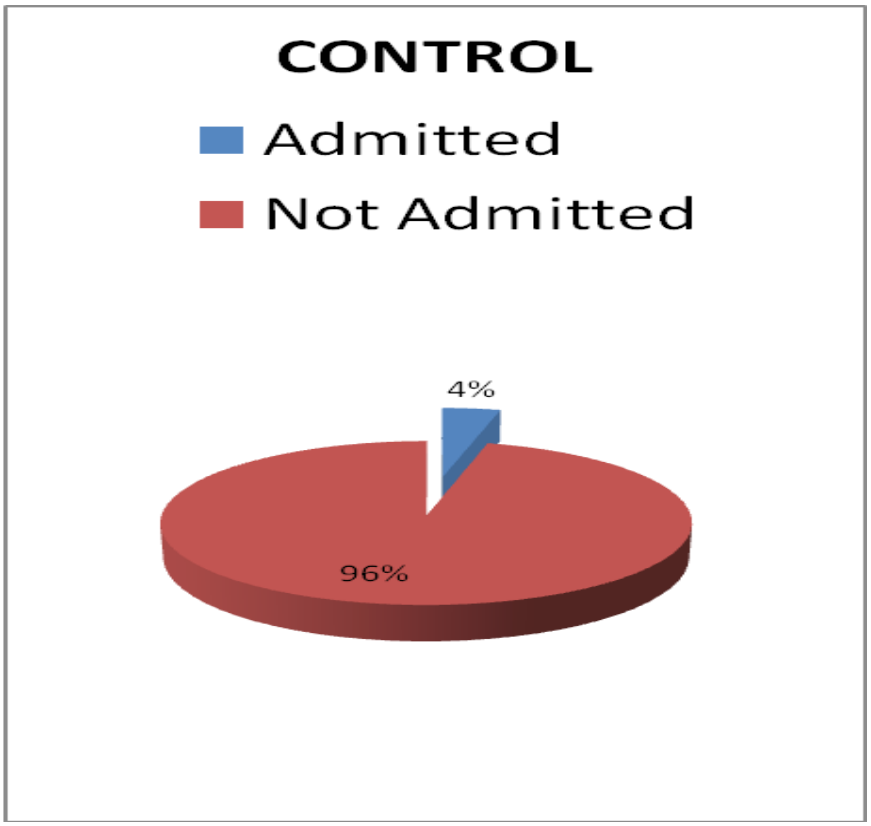
(Table14)

Admission Status	Case		Control	
	Number	Percentage	Number	Percentage
Admitted	78	78%	4	4%
Not Admitted	22	22%	96	96%

p value < 0.05 → significant.

The number of neonatal admissions in the case group clearly outnumbered the control group.78% of the babies in the case group were admitted whereas only 12% of the babies in the control group were admitted.


CASE
Admitted
Not Admitted
22%
78%

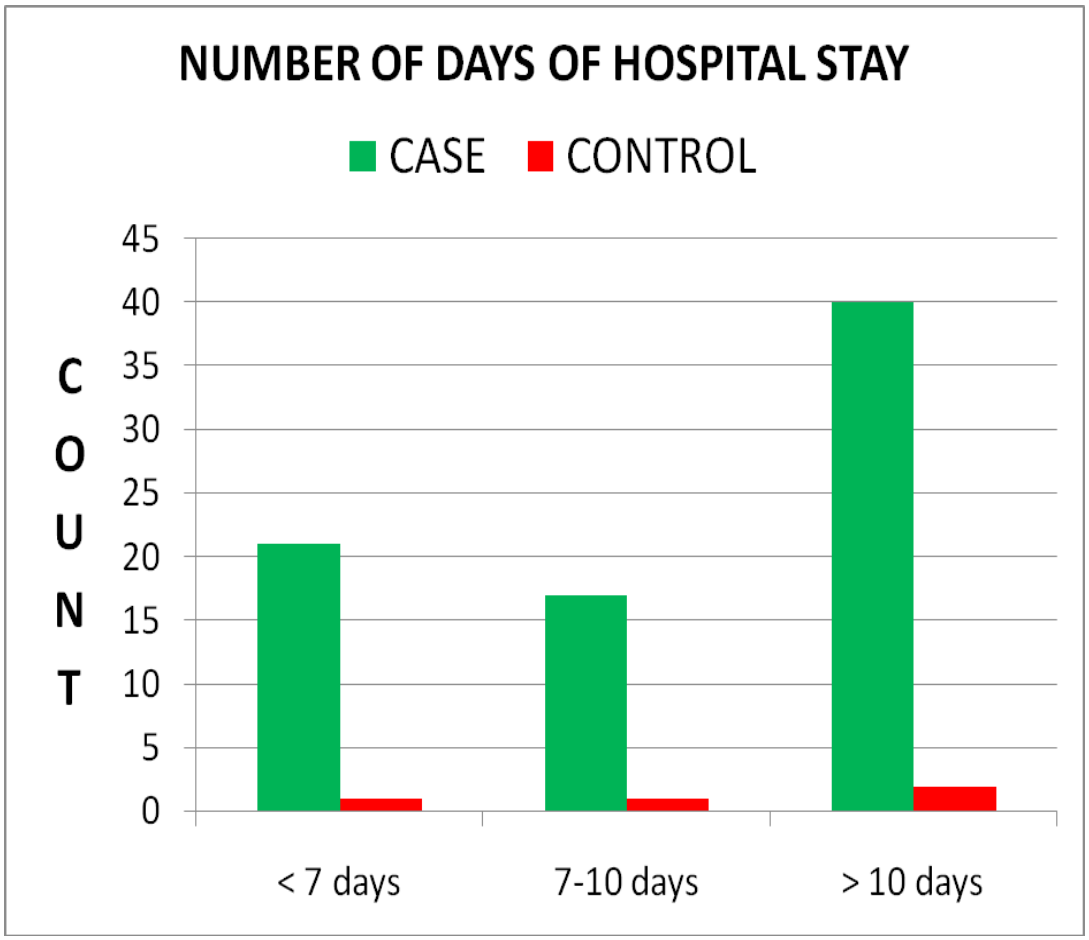

CONTROL
Admitted
Not Admitted
4%
96%

Number of days of hospital stay (Table15)

Number of days of hospital stay	Case		Control	
	Number	Percent-age	Number	Percent-age
<7 days	21	21%	1	1%
7-10 days	17	17%	1	1%
>10 days	40	40%	2	2%

Apart from the number of admissions being high in the case group, the number of days for which they were admitted also increased in the case group. 40% of the cases

had a hospital stay of more than 10 days, in comparison to 2% in the control group.



DISCUSSION AND ANALYSIS

The number of post-natal mothers originally interviewed and examined for the study was 253, out of which 31 were excluded due to the presence of vaginal/cervical infections and 22 were excluded as they had urinary tract infection. Finally 200 women were recruited for the study.

In the present study the sample size was slightly larger (200 subjects) when compared to the study conducted in North Carolina by **Offenbacher et al in 1996** [14] which consisted of 124 post partum women.

The mean age of the women in this study was about 24 years which was similar to the **North Carolina study** in which it was 22 years.

In this study 3% of the cases was under the age group of 15-20 years, 20% in the age group of 21-25, 65% in the group from 26-30%, 6% in the age group 31-35 and only 1% in the above 35 group. Though ages less than 18 years and more than 35 years are known risk factors for preterm labour relatively fewer cases belonged to these age groups in our study.

Among the controls too there was a similar age distribution with 2% of the subjects under 20 years, 63% in the age group of 21-30, 31% in the age group from

26-30 and finally 3% in the age group between 31-35. There was no subject above 35 years amongst the control group.

Both among cases and controls the maximum number of patients were in the 21-25 years age group. This represents the average child bearing age of the women in our country.

The periodontal examination was carried out within 72 hours post delivery coinciding with a study conducted by **Mroliterno et al in 2005** [43] as this indicates the previous disease condition and ensures the mothers were recruited prior to their discharge from the hospital.

In the present study the risk factors associated with preterm birth such as a low socio-economic class are in concordance with those reported in several other studies.

Though previous studies that link preterm labour and periodontitis such as the study conducted by **Offenbacher et al in 1996** [14] and **Mroliterno et al in 2005** [43] showed no such association, in the present study the women in the case group were from a lower socio-economic background and were less educated than the women in the control group.

Among the cases 93% belonged to Class V, 4% to class IV and only 3% to Class III. In the controls 72% belonged to Class V 16% to class IV and 12% to Class III. These values were found to be statistically significant. An analysis of their educational

qualification showed that among the cases 10% were illiterates, 71% had primary school education, 16% high school and 3% plus two education. There were no graduates in the case group.

In contrast in the control group the percentage of illiterates were only 2%,19% had primary school education and as much as 56% and 22% had high school and plus two education respectively. There was one lady who was a graduate among the control group. Once again these values attained statistical significance with a p value of less than 0.05.

Education is an indicator of awareness among the people and also represents their life style modification. In this study the controls had a better educational background than the cases .This could be the reason for better oral hygiene in them and consecutively a lesser incidence of periodontitis.

As to parity distribution the number of primipara were more than multipara in both cases and controls. Similarly in all parity groups there was an almost similar number of cases and controls. Hence in this study parity did not seem to affect the incidence of preterm labour.

Among multi para a previous bad obstetric history in the form of spontaneous abortion and previous preterm delivery had an increased risk for preterm birth in the subsequent pregnancies.

It is known that previous history of pre-term labour is one of the risk factors for a subsequent pre-term birth [46].

In the present study also previous preterm birth seemed to be more common amongst the cases than in the controls. 18.6% of the mothers in the case group had a previous preterm labour whereas < 1% of the mothers in the control group had such a history.

In a study on Periodontitis and pre-term labour conducted by **Mokeem et al in 2004** [40] consisting of 30 cases (infants < 37 weeks) and a daily random sample of 60 controls (infants > 37weeks), the risk of pre term labour remained high with increasing periodontal disease (odds ratio [OR] 4.21, 95% confident interval [CI] 1.99-8.93). As in the present study a history of previous pre term labour was more among cases than controls (p-value = 0.006).

It is not known why a woman with a previous pre term labour is at a higher risk to have a subsequent one. It is possible that in a woman with a positive history of pre term labour; the cause of the subsequent one may be the same factor that caused the previous.

In the present study there were eight cases but only one control that had a history of previous preterm birth. This was statistically significant with a p value<0.05. An interesting observation made was that two of these eight ladies in the case group with a

history of previous preterm labour had severe periodontitis. This could imply that the previous preterm births would also have been the consequence of periodontitis.

Analysis of the periodontal disease in the mothers in the case group and the control group clearly showed that there was a significantly greater number of mothers who had generalized chronic periodontitis (n=19) in the case group than in the control group (n=5).

Over the years in the various studies linking periodontitis and pre term birth a wide array of definitions have been used to describe and quantify the severity of periodontitis.

Offenbacher et al. in 1996 [14] stated that periodontitis corresponded to an average clinical attachment loss (CAL) of ≥ 3 mm at ≥ 60% of the examined sites.

Lopez et al in (2002) [16], similar to our study, examined all the teeth that were present in the dental arch and considered those mothers who had at least four sites with probing depth ≥ 4mm and CAL ≥ 3mm to be suffering from periodontitis.

In the present study in addition to this a category of mild periodontitis with and CAL < 3mm and severe periodontitis with CAL > 5mm was defined.

Table 9 clearly shows that among the 19 mothers with periodontitis in the case group 3 had mild periodontitis, 14 had moderate periodontitis and 2 had severe

periodontitis, whereas in the control group all the 5 mothers with periodontitis had the mild form. Thus there was relatively more number of women suffering from periodontitis in the case group than in the control group.

The results of the present study showed that the adjusted Odds Ratio (OR) for the association between periodontitis and preterm labour was **4.78 (95%CI=1.493-12.7).**

This association is in concordance with that which was originally suggested by **Offenbacher et al in 1996** [14] and confirmed in further studies carried out in United States (**Offenbacher et al 1998**) [37], Chile (**Lopez et al 2004**) [16], Thailand (**Dasanayake et al 1998**) [20] and Hungary (**Radnai et al 1998**) [53], with ORs varying between
3.5 and 7.9.

As is seen from table 10 the mean geastational age at delivery in the case group was 33.9 (±2.1) weeks and 38.1 (±1.3) weeks in the control group.

Goefpert et al in 2004 [52] conducted a case-control study in which, periodontal assessment was performed in 59 women who experienced an early spontaneous preterm birth at less than 32 weeks of gestation, in a control population of 44 women who experienced an uncomplicated birth at term (> 37 weeks). Severe periodontal disease was defined as a clinical attachment loss (CAL) of > 5mm at any one site. They found that severe periodontal disease had a significant association (p value=0.02) with

spontaneous preterm birth at less than 32 weeks of gestation. 49 women in the case group had severe periodontitis compared to 30 women in the control group.

This was similar to our study wherein those mothers with severe periodontitis delivered the most preterm infants with GA=30.5 weeks (95% CI=24.1-36.8). The lesser number of cases of severe periodontitis (n=2) in our study could be due to the more rigorous definition of severe periodontitis used (i.e., probing depth> 3mm at 4 sites with CAL>5mm at the same sites).

Further more in the present study the gestational age at delivery was found to be inversely proportional to the degree of periodontitis (Table 12). Those with moderate periodontitis delivered at a mean GA of 33.35 weeks (95% CI=32.5-34.1). In the case of mild periodontitis
it was 35.6 weeks (95%CT=34.2-37.1).

The CRP levels were higher in the cases than in the controls. Also the CRP levels were found to increase with increasing severity of periodontitis. The mean CRP level was 1.74 mg/l in pregnant women with periodontitis and 1.06 mg/l in those with no periodontitis. It was found to assume statistical significance with a p value < 0.05.

This was consistent with the study conducted by **Dr. Pitiphat et al in May 2006** [28] where the median CRP level was 2.23 mg/l (interquartile range: 0.74 to 4.14) in pregnant women with periodontitis and 1.46 mg/l (interquartile range: 0.71 to 3.58) in

those with no periodontitis.

As has been previously quoted evidence supporting the association between periodontitis and CRP is based mostly only on studies in men and non-pregnant women. There are only a few studies of periodontitis and CRP in either pregnant or post-natal women. This is an important area for study because systemic inflammation plays a major role in the pathogenesis of preterm delivery. CRP has been associated with adverse pregnancy outcomes, including preeclampsia, intrauterine growth restriction, and preterm delivery. Chronic infections like intrauterine infection and chorioamnionitis are linked to both preterm birth and elevated CRP levels. Therefore, CRP might be a plausible mediator of the association between periodontitis and adverse pregnancy outcomes.

In the present study those with severe periodontitis had the highest elevations of CRP-4.05mg/l (95%CI=3.1-4.9) (refer Table 9).The ones with moderate periodontitis had a mean value of 2.7mg/l (95%CI=2.5-2.9). In the case of mild periodontitis it was only 2.1mg/l (95%CT=2.04-2.21). The high CRP levels seen in severe periodontitis could well be the cause for the preterm births taking place at a lesser gestational age.

As can be expected, in the control group the mean weight at birth was 3.07 kg and in the case group it was 2.07kg. Amongst the cases, 7% babies weighed ≤ 1500 gm and two of these weighed only 980 gm. 29% of the babies had weight in the range of 1.6-2kg and 64% were in the range between 2.1-2.5 kg. There were none with birth weight

> 2.5kg (refer Table 13).There was significantly higher frequency of babies considered small for gestational age (SGA) in the case group.

In the control group 6% had birth weight >3.5kg, 53% of the babies were in the range of 3.1-3.5, 29% in the range of 2.6-3, 10% of the babies had birth weight between 2-2.5and just 2% had birth weight < 2kg. These two babies had severe Intra Uterine Growth Restriction (IUGR).

It was also seen that the mean birth weight also decreased with increasing severity of periodontitis (refer Table 13). In the severe periodontitis group the mean birth weight was 1.2kg (95%CI=-1.3, 3.7), in the moderate periodontitis group the mean birth weight was 1.8 kg (95% CI=1.6, 2.0) and in the mild form of the disease it was 2.2kg (95%CI=1.7-2.6). The values were most significant for severe periodontitis with the p value well below 0.05.

Table 14 shows the neonatal admissions, whereas 78% of the babies in the case group were admitted, amongst the controls only 4% were admitted.

The number of days of hospital stay was >10 days in 21% of preterm cases, 7-10 days in 17% and <7 days in 40% (refer Table 15). As the number of hospital admission increases it is more of an economic burden to the family and society. By decreasing the

number of cases of periodontitis a significant proportion of preterm births and low birth weight could be averted. This would in the long run help to boost the country's economy and would also reduce the prevalence of cerebral palsy and other effects linked to low birth weight and prematurity.

Thus, until recently only cervical/vaginal infections were thought to be capable of triggering inflammatory reactions leading to preterm labour. Data now supports the hypothesis that an inflammatory response to a distant infectious foci like periodontitis is also capable of causing preterm labour.

Our study supports this hypothesis with the odds ratio for developing preterm labour in a woman with periodontitis being 4.78.These findings indicate that it is in the patients best interest to include periodontal evaluation as a part of obstetrical and pre-natal care. Individuals with significant pathology could then be offered treatment likely to reduce the incidence of pregnancy complications. Furthermore oral health could be included in pre-conceptional counseling so that a lady could begin her pregnancy with a lower risk for preterm labour.

Obstetricians are at the obvious starting point to implement an interdisciplinary protocol to prevent preterm delivery. For the majority of individuals affected with periodontitis, the condition is symptom-free until the disease is more advanced. Therefore there is the need for medical personnel to increase the awareness among pregnant women [34]. The American Academy of Periodontology recommends that

every woman be referred for an oral examination early in pregnancy. The obstetrician should question every woman on symptoms of periodontitis such as bleeding or swollen gums. Those women who have had a preterm labour should be referred to identify and rule out for a potential role for oral inflammation in pregnancy.

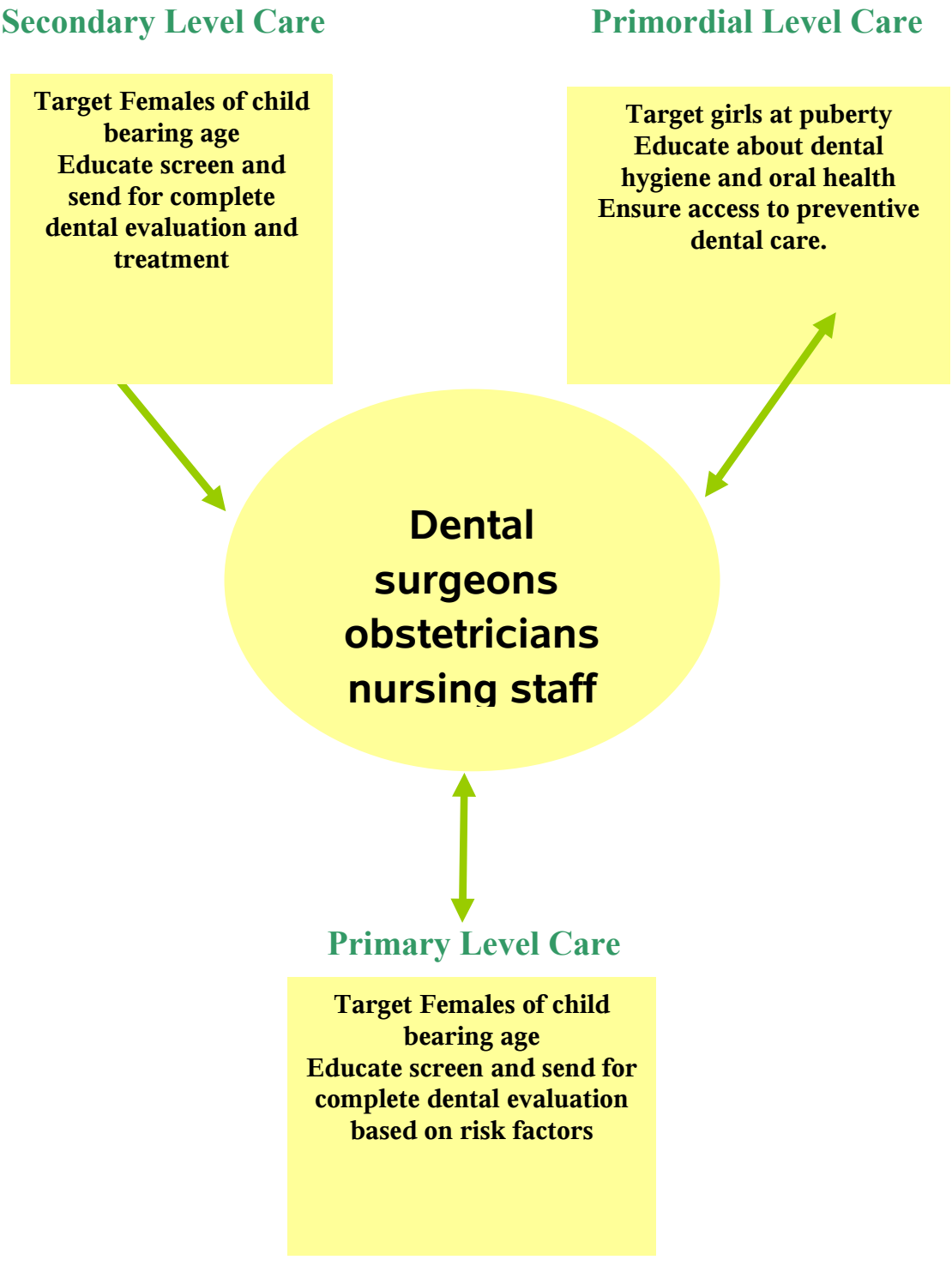
Importantly, the present study also provides evidence to support collaborative initiatives among health care providers in medicine and dentistry.

Dentists and dental hygienists can consult with obstetric specialists throughout a patient's pregnancy to monitor both her periodontal condition and CRP levels, as well as support the need for regular, comprehensive prenatal care. The physician, in turn must understand the dental professional's role in controlling the inflammatory process which can impact the systemic health of the pregnant woman.

This approach may allow for pregnant women with elevated CRP levels to be classified as "high risk," and thus evaluated more carefully for evidence of adverse oral health and pregnancy outcomes.

Thus both dentists and obstetricians have an obligation to collaborate and co-ordinate efforts to deliver adequate care for women of child bearing age. This transdisciplinary approach though presents unique challenges would reap rich dividends if implemented.

Interdisciplinary Preventive Care



SUMMARY AND CONCLUSION

The study concludes that there is a significant association between periodontitis and pre term labour. The odds ratio for developing pre term labour in women with periodontitis was calculated to be 4.78.

Moderate and severe varieties of periodontitis were associated with lower gestational age at delivery when compared to mild periodontitis.

The CRP value was found to be elevated in those with periodontitis when compared to those women who were periodontally healthy.

There was significant correlation between increased CRP levels, increasing severity of periodontitis and prematurity.

CRP could be the biologic mediator between the periodontiotis and preterm labour.

The present study provides evidence to support the need for collaborative initiatives among health care providers in medicine and dentistry.

PERFORMA

Name : LMP:

Age : EDD:

I.P Number:

Socio-Economic Class:

Educational Qualification:

Date of delivery:

Gestational age at delivery:

Gestational age as per 1st trimester ultra sonogram:

Past Obstetric history:

H/o excessive foul smelling vaginal discharge:

H/o Diabetes/heart disease/ hypertension/PIH/multiple gestation

H/o expulsion of still born foetus:

H/o any tooth extraction in the past:

H/o toothache/ bleeding from gum:

Details about the baby:-

1. Information about the maturity of the baby obtained from post–natal evaluation of baby by a pediatrician:

2. Birth weight of baby:

3. Admission status of the baby:

4. Number of days of admission:

Examination

General Examination:

Temperature:

Pulse:

Blood Pressure:

Per abdomen Examination:

Speculum Examination:

Biochemical and Microbiological Investigations:

Hb %:

Blood Sugar:

Blood Urea:

Sr creatinine:

CRP Level:

Urine for Culture:

KOH whiff test for Bacterial Vaginosis:

Wet Mount Examination:

Results of Periodontal Examination:

Periodontitis: Present/Absent

If present: Localized/Generalized

Degree of Periodontitis: Mild/Moderate/Severe

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